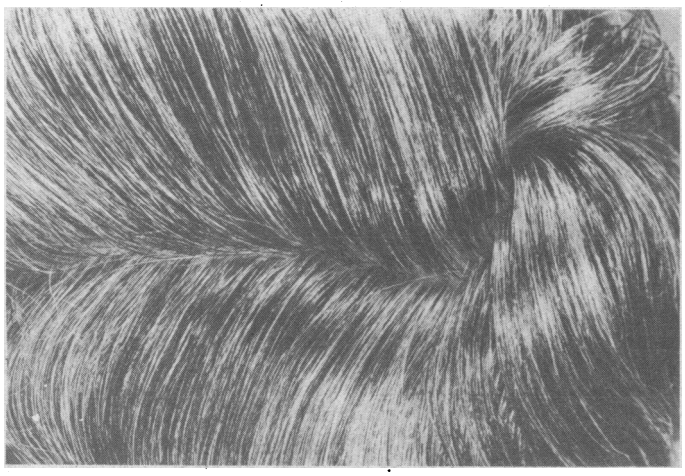
Reversal of premature hair greying in adult coeliac disease

There is no known association between premature greying of the hair and the subsequent diagnosis of adult coeliac disease. I report two cases in which the hair colour turned completely during the third decade and remained unchanged until adult coeliac disease was diagnosed at least 20 years later. In both cases a reversal of the colour change began a few weeks after starting a gluten-free diet.

Case reports

(1) The patient, a man, had had dark brown hair until the age of 25. Discoloration progressed until at the age of 30 the scalp hair was snow white. Other body hair was unaffected and he remained in good health. At the age of 53 he was admitted to hospital after an accident and was found to be anaemic. The results of investigations, including jejunal biopsy, were consistent with adult coeliac disease. Institution of a gluten-free diet led to resolution of the anaemia. Within three months of starting the diet his hair was noticeably darker at the temples. Within five years his hair was quite



Hair banding related to clinical relapse of adult coeliac disease.

dark but with streaks of grey, appropriate for his age of 58. Later his bowel action of two soft stools daily changed to one firm stool daily, but he could not remember if his bowel action had ever been different in his youth. A deliberate gluten meal caused diarrhoea, which settled over six weeks, and the freshly emerging hair became light grey once more, returning to the darker colour as the diarrhoea settled. A second gluten meal caused a repetition and obvious bands could be seen (figure).

(2) The patient, a woman, had lost her dark brown hair colour during her late thirties and became completely white by the age of 43. She had recurrent iron-deficient anaemia which was attributed to menorrhagia until after the menopause, when she was investigated. The findings, including jejunal biopsy, suggested adult coeliac disease. Within a few weeks of starting a gluten-free diet at the age of 58 her hairdresser commented that her hair was growing out darker, and by the age of 63 its appearance strikingly resembled that of the first patient. The hair change was restricted to scalp hair and there was no alteration of bowel habit, but she felt much better.

Comment

The hair appearance may change in childhood protein deficiency states[1] and in inborn errors of amino-acid metabolism.[2] The reason for the change is unknown but perhaps abnormal amino-acid concentrations in the body fluids produce abnormal protein. Patients with phenylketonuria have an abnormally high phenylalanine content in their imperfectly pigmented hair,[3] and patients with kwashiorkor have a deficiency in the number of melanosomes in the cornified hair[4] and a reduction in the amount of sulphur-containing protein with corresponding depigmentation.[5] The amino-acid content of hair samples from the patient in case 1 was analysed, comparing dark and light zones. There was no difference in the concentration of 17 standard amino-acids and the values obtained were within normal ranges.[3] Possibly minor differences were not shown and more subtle changes would be expected in coeliac disease than in much more florid deficiency states. Also the sulphur-containing amino-acids are difficult to assay accurately[5] and it is these that are concerned in maintaining the three-dimensional structure of protein fundamental in the determination of colour.

Although the mechanism of the hair colour changes in these two cases is not clear its connection with gluten sensitivity is undoubted. Perhaps this type of premature greying is either the presenting sign of adult coeliac disease or is a coincidental predictor of its later occurrence.

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[2] Vellan EJ, Gjessing LR, Seip M. Hair amino-acids in cystinosis, homocystinuria, Fölling's disease and tyrosinosis. *Acta Paediatr Scand* 1969; **58**:287-9.

[3] Van Sande M. Hair amino acids: normal values and results in metabolic errors. *Arch Dis Child* 1970;**45**:678-81.

[4] Sims RT. *An introduction to the biology of the skin.* Oxford: Blackwell Scientific Publications, 1970:387.

[5] Pollitt RJ, Stonier PD. Proteins of normal hair and of cystine deficient hair from mentally retarded siblings. *Biochem J* 1971;**122**:433-44.

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Acute renal failure and myopathy after treatment with aminocaproic acid

Epsilon aminocaproic acid (aminocaproic acid) is increasingly being used to inhibit fibrinolysis in subarachnoid haemorrhage. We report a case in which acute massive muscle necrosis and acute renal failure occurred after treatment.

Case history

A 20-year-old nursery nurse was admitted to hospital with a typical subarachnoid haemorrhage confirmed by lumbar puncture. Carotid angiography and CT scan showed a large right-sided arteriovenous malformation. She was given aminocaproic acid 30 g daily for seven weeks. After six weeks' treatment myalgia, haematuria, and proteinuria were noted. Over the next week this progressed to profound muscle weakness and severe respiratory impairment. Despite previously normal renal function she developed acute oliguric renal failure necessitating haemodialysis.

Examination showed gross generalised flaccid weakness, and the muscles were so swollen and tender that physiotherapy was initially extremely difficult. Serum urate concentration was 0·57 mmol/l (9·6 mg/100 ml), sodium concentration 116 mmol/(mEq)/l, urea concentration 44·0 mmol/l (265·0 mg/100 ml), potassium concentration 5·4 mmol(mEq)/l, and creatinine concentration 610 μmol/l (6·9 mg/100 ml). The table shows the serum creatine kinase and aspartate transaminase activities during weeks 1-5

Serum creatine kinase and aspartate transaminase activities during weeks 1-5

Week:	1	2	3	4	5
Creatine kinase (IU/l)	22 987	20 504	1271	310	73
Aspartate transaminase (IU/l) ..	242	139	104	10	35

A muscle biopsy specimen showed acute, massive muscle necrosis with no evidence of polymyositis, polyarteritis, or any occlusive or thrombotic vessel disease. Results of other haematological investigations, tests for collagen diseases, virological studies, and chest radiography were normal.

After four weeks renal function recovered and the patient could sit upright with support. Three months later muscle power in all limbs was almost normal.

Comment

This report illustrates two grave consequences of treatment with aminocaproic acid, each of which is potentially lethal. The onset of acute massive myonecrosis six weeks after beginning treatment